Zapped for safety. Advocates say that irradiating food offers real protection against microbes, but some still worry about the safety of the procedure.

Irradiation will pasteurize, not sterilize, meat, say food safety specialists. The growth of harmful bacteria, such as Salmonella, Campylobacter, and *E. coli* O157:H7, and other microorganisms that spoil food but pose no harm will be sharply retarded because there will be fewer of them.

Irradiation is by no means the final answer to food safety concerns, however, experts say. "It's a complement to the other practices. The more safety barriers you have, the less likely you're going to have an unsafe product," says George Pauli, director of the division of product policy at the FDA's Center for Food Safety and Applied Nutrition in Washington, DC. Pauli and other authorities emphasize that irradiation doesn't relieve consumers from their responsibility in handling food safely. While he cannot cite exact figures, Morris Potter, an epidemiologist with the CDC says irradiation should "vastly improve the level of food safety."

Critics, however, claim that food irradiation is not necessary and may even harm food. Michael Jacobson, executive director of the Center for Science in the Public Interest, a nonprofit education and advocacy organization in Washington, DC, said in a 2 December 1997 statement issued by the center that there are other, cheaper methods, such as steam pasteurization, to make sure foods are safe. In the statement, he objected to the expected added expense of several cents per pound to cover the cost of irradiation equipment, ad added that "the meat and poultry industry should invest in new technologies that create clean products with less expense."

Food and Water, Inc., a Vermont-based activist group, argues that irradiation lowers the nutritional value of meats. Food specialists agree, but counter that the loss is no greater than when meat is cooked. Food and Water also questions the safety of the radiation process, arguing that it may cause illnesses and chromosome damage in those that eat it. But Pauli dismisses that possibility. He says the FDA approved irradiation under a legal standard that it would cause no harm, according to "competent scientists."

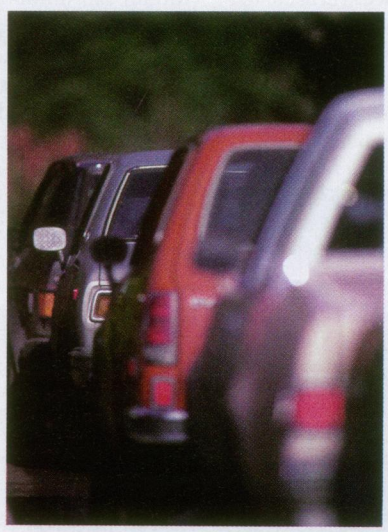
How irradiated meat will fare at the counter is uncertain. Opponents and proponents each have polls supporting their view on whether the public approves or disapproves. But consumers will know what they are getting, since a label indicating the meat has been irradiated must appear on the package. The label, however, is too small, according to Food and Water.

In the meantime, there will be efforts to make sure that conditions are optimal for meat irradiation, notes Doyle. One problem to be solved is to make sure "off" flavors don't develop. "The fattier the food, the more likely it is that off flavors will develop," he notes. He also points out that ground beef, which can be 20–30 % fat, may pose a challenge.

Genes and Ozone

New research suggests that whether or not a person reacts to toxic levels of ozone in the air depends upon their genes. If the animal studies that support this association are confirmed in humans, this newest example of the interaction between genes and the environment could have untold implications for industry, insurance, and health.

Researchers believe that knowing they are susceptible could help people protect themselves on bad ozone days, and they hope that clinical genetic therapies might also eventually be developed. Others say information on susceptibility could lead to stricter government regulation of air quality to reduce nitrogen oxides from car exhausts, which combine with oxygen and sunlight to form ozone.



Ozone culprits. New research shows that not just exposure but also genetics may play a role in susceptibility to the effects of ozone created when sunlight and oxygen mix with auto exhaust.

Yet another view is that such information could create a subpopulation of people at risk for discrimination on the basis of their genetic makeup. "It could be a sticky issue if the gene ran in certain ethnic or racial groups, or if disclosure of the gene could risk insurance coverage," says pulmonologist Jeffrey Drazen of the Harvard Medical School in Cambridge, Massachusetts.

"This could become quite a societal issue," acknowledges Steven Kleeberger, one of the scientists who reported the link in the December 1997 issue of *Nature Genetics*. Kleeberger, a researcher at the Johns Hopkins University department of environmental health sciences, predicts that, given genetic susceptibility, the health effects of ozone will become even more of a regulatory and political issue in the future. The findings of Kleeberger's research, along with those of a second study by George Leikauf and colleagues from the University of Cincinnati that were published in the same issue, move the field of air pollution genetics solidly forward. Now, scientists don't talk about if such susceptibility genes are identified in humans, but when.

Both studies used strains of inbred mice with differing responses to ozone. Kleeberger's team selected one strain of mice that was resistant to ozone and one strain that was very responsive. They crossbred the groups, then bred the groups' offspring to select for expression of genes on chromosome 11 and chromosome 17 that control responsivity to ozone. The Cincinnati researchers also found a locus on chromosome 11 that broadly overlapped with Kleeberger's area, indicating the two teams may be honing in on the same gene.

There was significant activity in the segment on chromosome 17, and in searching the mouse genome database for this chromosome, Kleeberger and his team identified several candidate genes that may be causing the activity. One, the tumor necrosis factor alpha (*TNF-α*) gene, seems a highly logical candidate, Kleeberger says. TNF is a pro-inflammatory cytokine that influences genes in the immune response cascade. To test their hypothesis, Kleeberger's team treated the susceptible strain of mice with antibodies that

neutralized the TNF-α protein. The reaction of these mice to ozone was similar to that of the resistant mice. Although Kleeberger calls this evidence "intriguing," it is not proof that *TNF-α* is the controlling gene in ozone susceptibility, he says. More linkage and physical map studies are needed to identify what he suspects may be a bevy of major and contributing genes associated with differing susceptibilities to ozone.

Indeed, Aravinda Chakravarti, a professor of genetics and medicine at Case Western Reserve University in Cleveland, Ohio, questions the use of mice as surrogates for how ozone affects human biology. The inbred mice used in the studies surely have less genetic variation in their reaction to ozone than that found in humans, he says, and moreover, humans metabolize oxygen differently.

But Kleeberger points out that the "mouse represents a unique model to study genes in their most simple representation." Michael Blaese, chief of the clinical gene therapy branch at the National Human Genome Research Institute in Bethesda, Maryland, says it makes sense that *TNF-α* may be in some way linked to ozone susceptibility because it is involved in so many important biological activities.

Even so, Blaese believes that any effort to clinically correct or repair susceptibility genes will be a long time in coming because of the inherent difficulties of gene therapy. More likely, he says, researchers will search for clinical ways to interfere with the protein encoded by the gene.

This work offers hope to millions of asthmatics, says Sharon Hipkins, director of programs and policy at the Asthma and Allergy Foundation of America in Washington, DC. It highlights "clinical recognition that there probably is a familial trait that mediates reaction to high ozone levels and that, likely, many asthmatics are affected," she says. But Kleeberger says that asthma is a very complex disorder with a number of different phenotypes and that the association between ozone and asthma susceptibility is not completely understood.

"What is most interesting about these studies is that we have always viewed air pollution as having adverse effects on society as a whole," says Drazen. "Like reactions to medications, we are learning we might be able to identify selected individuals who are affected."

EHPnet

Mad Cow Media

"Mad cow disease," the common name for bovine spongiform encephalopathy, or BSE, is alone enough to generate concern in anyone who consumes the daily news along with his hamburger. When the disease broke out among humans in 1996, there was panic among the public, who feared that eating beef would result in death from the "brain-rotting" disease. To help combat such public hysteria, the private charitable Sperling Foundation established the Official Mad Cow Disease Home Page, located at http://www.mad-cow.org/. The page is a repository for over 3,650 articles on BSE and related pathologies, and is updated twice a week (hourly during breaking events). News clips from a vast array of sources are warehoused and cataloged according to topic.

The Official Mad Cow Disease Home Page is managed by Tom Pringle, scientific director of the Sperling Foundation's Creutzfeld–Jakob disease (CJD) program (CJD is commonly known as the human equivalent of BSE). Says Pringle of the site, "Basically, my ambitions for the site were to create a new paradigm for human disease research, based on unique aspects of the Web such as unlimited available space and essentially no distribution costs. This allows much more depth in terms of articles archived, much more frequent updating than a fixed journal, room for supplemental commentary and clarification by appended correspondence with scientific authors, and opportunity for value-added background links."

The home page features an extensive list of links to articles divided by topic into 10 categories: General News; Prion Molecular Biology; Victims; Prion Research; BSE in Blood, Milk, Meat; How Beef is Made; Alzheimer's and CJD; Other Cow-to-Human Diseases; Epidemic; and Mad-Cow Scientists. These articles, culled from the lay press, present a chronicle of the development of the mad cow disease scare and research that is being done to identify the source of this alarming malady. The home page also lists links to several different information resources, along with a site search engine and technical e-mail correspondence and sharing of news with Pringle.

Over 200 links to different online journals, search engines, databases, and other resources are listed from the home page under The Best Links. Some of these links are for general Internet reference areas, while others are specifically relevant to the topic of BSE, such as the Food Safety and Inspection Service's listing of food recalls from 1990 through 1998. This page also contains several links to online molecular biology tools, such as the University of Illinois's NCSA Biology Workbench.

The Real Science link on the home page leads to a list of links to scientific papers on the study of BSE. The papers are grouped on the page by topic, with each link leading to an abstract and citation for the original paper. This link also includes reviews by Pringle of current research and tables—updated regularly—of known mutant strains of BSE. Pringle posts his own research as well, saying, "I strongly believe that this is the future of biomedical research and an ethical mandate in the human disease context."

The 3D Interactive Prions link allows visitors to view two- and three-dimensional renderings of the prion protein molecule. Finally, the home page includes a link to the Fundraising and Support for CJD Victims page, a list of support groups for CJD sufferers and their families.